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THE AMBULATORY SEMI-POIKILOTHERMIC DOG
(Poikilothermic Against Cold)

by

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from

Physiology Department

Submitted

10 June 1952

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Abstract;

Discussion of ambulatory poikilothermic homeotherms to be used as a tool for studying the effects of thermal variables uncomplicated by superimposed neurogenic regulatory influences, and as a background basis for assaying or evaluating partial impairment of thermo-regulatory functions.

Ambulatory semi-poikilothermic dogs were prepared by selective thermo-coagulation of the posterior hypothalamic gray. They were only semi-poikilothermic to the extent that all ability to regulate against cold was absent, but they retained adequate regulation against heat.

ARMY MEDICAL RESEARCH LABORATORY

FORT KNOX, KENTUCKY

REPORT NO. 84

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THE AMBULATORY SEMI-POIKILOTHERMIC DOG* (Poikilothermic Against Cold)

*Subtask under Environmental Physiology, AMRL Project No. 6-64-12-023, Subtask, Experimental Physiological Deficits.

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THE AMBULATORY SEMI-POIKILOTHERMIC DOG*
(Poikilothermic Against Cold)

by

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10 June 1952

*Subtask under Environmental Physiology, AMRL Project No. 6-64-12-028, Subtask, Experimental Physiological Deficits.

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ABSTRACT

THE AMBULATORY SEMI-POIKILOTHERMIC DOG (Poikilothermic Against Cold)

OBJECTIVES

1. To prepare an ambulatory poikilothermic homotherm to be used: (a) as a tool for studying the effects of thermal variables uncomplicated by superimposed neurogenic regulatory influences, and (b) as a background basis for assaying or evaluating partial impairment of thermo-regulatory functions.
2. To ultimately associate neurogenic thermo-regulatory functions with specific anatomical structures.

RESULTS

1. Ambulatory semi-poikilothermic dogs were prepared by selective thermo-coagulation of the posterior hypothalamic gray. They were only semi-poikilothermic to the extent that all ability to regulate against cold was absent, but they retained adequate regulation against heat.
2. These preparations were ambulatory in that they were oriented with their surroundings sufficiently to eat and drink spontaneously, maintained good nutrition, groomed themselves and showed no motor or postural deficits.
3. The animals prepared thus far have exhibited a spontaneous pathological susceptibility to rage which is a troublesome factor in using the preparations as laboratory tools.

CONCLUSIONS

1. The neurogenic regulation of body temperature against a cool or cold environment is dependent upon neural elements located within the posterior hypothalamic gray.

2. Regulation against a warm or hot environment is not eliminated when the posterior hypothalamic gray is selectively destroyed.

3. Posterior hypothalamectomy does not eliminate the animal's orientation with its surroundings, its ability to eat and drink spontaneously, produce obvious gastrointestinal or nutritional deficits, or impair its motor or postural mechanisms.

4. Such preparations are truly ambulatory but the presence of an increased susceptibility to rage limits their usefulness as laboratory tools.

RECOMMENDATIONS


1. A continuing effort should be made to prepare poikilothermic dogs devoid of troublesome personality traits.

2. The localization of the neural elements responsible for regulation against heat should be further delimited.


3. The anatomical basis for the striking changes in personality encountered in the experiments should be systematically investigated.

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THE AMBULATORY SEMI-POIKILOTHERMIC DOG (Poikilothermic Against Cold)

I. INTRODUCTION

The desirability of having a chronic poikilothermic homotherm available for evaluating the effects of thermal variables uncomplicated by superimposed regulatory influences has long been appreciated. Isenschmid and Schnitzler (1) in 1914, and Bazett and Penfield (2) in 1922, made decided efforts to maintain mid-brain rabbits and cats, respectively, to chronicity with the foregoing end point as a principal objective. Both teams of investigators failed to maintain their animals to long-term chronicity. The limiting factor in both instances appeared to be predominantly a matter of adequate nursing care.

The senior author, some years ago, became interested in determining the effects of destructive lesions placed in the hypothalamus, care being taken not to allow the tissue defect to infringe upon neighboring structures. The primary objective was that of determining if an animal's ability to maintain its body temperature near constancy was actually dependent upon hypothalamic tissue, as Ott (3) originally suggested in 1895 on the basis of puncture experiments, and as was clearly indicated by the brain-slicing experiments of Isenschmid and associates in 1914 (1). A secondary objective was that of determining if this approach would produce poikilothermic animals which would be ambulatory to the extent of their not needing the type of special nursing care which is entailed in maintaining brain stem preparations (4).

In a first group of experiments on cats (5), the entire hypothalamus was macerated by a small blunt probe. These animals exhibited no regulatory ability against an external cooling load but, surprisingly, the heat loss outflows remained intact and in addition were actually released from coordinated control. Chronicity was not obtained because in a matter of a day or two the animals deteriorated rapidly, usually being found dead the second or third morning after operation.

In a second group of experiments on dogs (6, 7), the hypothalamic gray was simply isolated from its dorso-caudal connections, most of this tissue being left undisturbed otherwise. Several such preparations were successfully maintained for months, i. e., to long-term chronicity. They exhibited a complete inability to prevent a lowering of body temperature when subjected to any sort of an external cooling load, yet they were able to combat a warm or hot environment in an

entirely adequate manner. This thermo-regulatory status was present immediately after operation and remained continually present with no appreciable tendency to lessen as long as the animals were kept alive.

In this instance chronic, semi-poikilothermic (against cold) preparations were obtained but they were not ambulatory in any sense. They presented nursing care problems; they were lethargic, were not oriented with their surroundings, did not eat or drink spontaneously, and death eventually occurred from aspiration or hypostatic pneumonia or similar complications.

The purpose of this communication is to describe a third group of experiments in which the primary objective was that of attempting to prepare a completely ambulatory poikilothermic dog which could be used as a "physiological test tube" for the study of the effects of thermal variables as well as for the further elucidation of the processes involved in the mechanisms of thermo-regulation. It was reasoned on the basis of previous experience that a successful selective destruction of the posterior hypothalamic gray should attain such an objective.

II. EXPERIMENTAL PROCEDURES

A. Animals.

Small female dogs, in general of the terrier type, ranging in weight from 6 to 10 kilograms were used. Previous to operation they were conditioned to laboratory and feeding regimens.

The daily diet was constant, consisting of 10 grams Purina Laboratory Chow and 10 grams frozen horse meat per kilogram body weight. Water was available in the cage continuously and if after operation there was a tendency not to drink the preoperative amount of water, this amount of fluid was given by baiting it with canned milk or meat juice.

B. Surgery.

The pituitary area was exposed by the Bailey and Bremer modification of Cushing's subtemporal approach. This consists in (a) a midline skin incision reaching from a point between the eyes cephalically to the midcervical level caudally, (b) a T-shaped incision through the temporal muscle with an appropriate retraction cleanly exposing the bone of the skull which lies underneath it, (c) removal of the exposed bone by drilling a hole with a trephine and enlarging it

with rongeurs, and (d) elevation and retraction of the temporal pole with a rigid and appropriately shaped spatula. The last procedure is carried out after the head has been rotated 90° from its normal position, that is, with the right side of the head uppermost and the left side undermost. The rotation and fixation of the head and operative field was accomplished by means of a Brodie dog head holder mounted to a mechanical manipulator. This procedure places the base of the brain in a direct vertical plane with the operator's vision. Under optimum conditions, which is usual, the optic nerves, optic chiasm, right internal carotid and its lateral branches, the right side of the pituitary stalk and pars anterior, the right oculomotor nerve, the ventral aspects of the mammillary bodies, the right cerebrospinal peduncle, the cephalic and ventral aspects of the pons, the interpeduncular fossa and the right aspect of the sella turcica are all clearly visible in a clean and dry field.

After the exposure is made, a needle electrode bent at a 120° angle is pressed through the pia in the midline at the cephalic aspect of the mammillary bodies. In this position, a spurt of coagulating current is applied. Subsequently, through the opening thus made in the pia, a small ball-electrode bent at a 90° angle is inserted in a midline position and 5 seconds of coagulating current set at 20 on a Bovie unit is applied. This electrode is then projected as deep as the long arm of the electrode allows (3 mm.) and the same procedure repeated. A second 5 seconds of coagulating current is then applied and during its application the electrode is pushed gently, slightly to the left and then to the right. These procedures are executed with a view of devitalizing all the posterior hypothalamic gray without infringing upon the thalamus, the hypophysis, the cerebrospinal tracts, the anterior hypothalamic gray, and the tissue lying caudal to the mamillothalamic tracts. A photograph of the medial aspect of a dog's brain is shown in Figure 1(b) in which such a selective lesion was successfully placed in a practice run.

Closure is made by replacing the temporal pole, approximating the edges of the dura and suturing the temporal muscle and skin. The temporal pole is placed back into its fossa, relaxed and without evidence of edema. No attempt is made to suture the edges of the dura.

C. Location and Extent of Hypothalamic Gray.

One gets the best impression of the location and extent of the hypothalamus by viewing the medial aspect of a brain that has been cut in half along the midline in a sagittal plane. A photograph of such a view of a dog's brain is shown in Figure 1(a). Note that the hypothalamus is bounded dorsally by the thalamus, ventrally by the

hypophysial stalk and optic chiasm, caudally by the mammillary bodies and cephalically by a line drawn from the anterior commissure dorsally to a point a few mm. ahead of the optic chiasm ventrally.

The markings of the hypothalamus, however, can be better appreciated by viewing Pal-Weigert sagittal sections through the hypothalamic area. A photograph of such a section through the plane of a mammillothalamic tract is shown in Figure 2(a) and one through the plane of a fornix in Figure 2(b). Note how clearly the gray of the hypothalamus is demarcated from the neighboring structures containing medulated fibers.

It is to be particularly noted that this gray extends caudally only to the cephalic border of the mammillothalamic tracts. The term hypothalamic gray is used in preference to hypothalamus for several reasons but primarily because of its greater specificity. It is important to point out that the term hypothalamic gray is not synonymous with the third ventricular gray; it includes both the medial and lateral components of the hypothalamus as they are generally delimited. The lateral hypothalamic gray extends laterally to roughly approximate the medial aspect of the cerebrospinal tracts.

It has recently become common practice to arbitrarily divide the hypothalamus into anterior, posterior, ventral and dorsal components. Thus, the fornix, as shown in Figure 2(b), is the focal point for dividing the hypothalamic gray into ventral and dorsal halves and into medial and lateral components. The division of the gray into posterior and anterior halves is strictly on an arbitrary basis as can readily be seen when looking at the photographs in Figure 2. In this connection, the photograph in Figure 1(b) illustrates the separation of the hypothalamic gray into these two halves as well as this can be done. Note that in this photograph the anterior hypothalamic gray remains intact whereas the posterior hypothalamic gray is completely and selectively destroyed.

D. Determining the Location and Extent of Tissue Defect.

The animals were terminated under full barbiturate anesthesia by opening the thoracic cavity, cutting the superior vena cava, and perfusing the body through a stab puncture in the left ventricle first with 0.9% saline followed by 5% formalin in 0.9% saline.

The brain was removed immediately, care being taken that the pituitary fossal contents, including the dura, remained attached to the hypothalamus without any disturbance in these attachments. The

brain was then placed in 10% formalin for 24 hours after which it was blocked appropriately and again placed in 10% formalin for an additional 24 hours or longer.

The block was subsequently processed and embedded in paraffin and sectioned at 20 μ . Usually two sets of sections were mounted in serial order taking every 20th section, one set being stained with a modified Pal-Weigert procedure and the other with cresyl violet or an equivalent cell stain. Photographs of Pal-Weigert sections showing the location of the tissue defect in representative experiments are shown in Figures 5, 8, 9, and 10.

E. Assay of Heat Regulating Abilities.

1. Regulation Against a Cooling Load: The preparation's ability or lack of ability to maintain body core temperature within the usual range of 38-39°C against a cooling load was evaluated in two ways. First, it was determined if it was necessary to house the animal in an incubated external environment. If so, then the "regulation interval" of Isenschmid and Schnitzler, namely the difference between the environmental temperature and body core temperature of the animal under basal conditions was determined. This was usually noted the first thing in the morning, that is 24 hours after food and after the long night period of relative inactivity. Secondly, the animals were abruptly subjected to a heavy cooling load (3-5°C) and the rate of fall in body core temperature (deep rectal or colonic) and the presence or absence of shivering were observed. The animal was always removed from the 3-5°C exposure when the body core temperature approached 28°C and placed in a room held in a 22-23°C range to determine to what extent the core temperature would rise spontaneously, thus determining semi-quantitatively the regulation interval at a lower ambient temperature range.

2. Regulation Against a Heating Load: The preparation's ability or inability to prevent an undue rise in body core temperature was tested by placing it abruptly into warm or hot rooms for a matter of a few hours or a few days. One test situation used was a dry bulb temperature of 35-37°C and a relative humidity near 30% with rapid air movement. This was considered a moderate heating load because unoperated dogs when quiet will tolerate this load without panting, cardiovascular adjustments obviously being adequate for necessary dissipation of heat. A second test situation was a dry bulb temperature of 37-39°C and a relative humidity near 30% with rapid air movement. This was considered a relatively heavy heating load since it always elicited panting, at least periodically, in the resting unoperated dog.

III. RESULTS

A. Total Absence of Cold-combatting Powers.

The permanent elimination of all abilities to regulate against any sort of cooling load has been attained in several instances. The data on the chart shown in Figure 3 illustrate the criteria upon which the complete absence of cold-combatting powers was based. In this figure deep rectal or colonic temperature curves of Dogs No. 28 and 35 are contrasted with the curve of an unoperated Dog 46 when they were simultaneously subjected to the same heavy cooling load. First note that it was necessary to maintain Dogs 28 and 35 in an environment of 30-31°C in order to keep their core temperatures at a 38°C range. Thus, the "regulation interval" of Isenschmid was 7 or 8°C at these (ambient and body core) temperature levels. Secondly, when they were abruptly exposed to an ambient temperature of 3°C, their body temperatures fell progressively in a straight line, reaching 28°C by the end of a three-hour exposure. There was no shivering. Thirdly, when the animals were removed to an ambient temperature of 22-23°C for a three-hour period, the core temperature remained stationary. Here the regulation interval was 5-6°C, one or two degrees less than at the temperature ranges which obtained previous to the exposure. As soon as the animals were returned to a 30-31°C environment, the body temperatures immediately began rising; having reached 38°C by the following morning.

The rate of fall in body core temperature varies in such non-heat regulating dogs depending upon factors such as (a) thickness of subcutaneous fat layers, (b) movements of the animal during the test and (c) the ambient temperature to which the preparation was subjected previous to the initiation of the test; particularly if the preparation has retained its regulating abilities against heat. The latter point is shown in an extreme manner in Figure 4, which illustrates graphically two different experiments on Dog 124. In the first experiment this dog was placed into a 4° ambient temperature directly after being housed in a room held at 28°C. The regulation interval was 7 or 8°C previous to the exposure to cold and the post exposure regulation interval was 6°C. Body core temperature dropped rapidly at the rate of 3.5°C per hour as was the case in Dogs 28 and 35 illustrated above. In the second experiment the animal was placed into the 4°C room directly after being housed for 24 hours in a 37-39°C room. The animal tolerated the mean 38°C ambient temperature for 24 hours adequately though the core temperature rose slightly above the usual level during the last 12 hours.

In being abruptly subjected to an ambient temperature of 4°C the body core temperature fell much more rapidly than in the previous instance, namely, 8°C per hour rather than 3.5°C. The explanation is obvious -- all heat dissipating avenues were fully activated. The regulation interval previous to being placed in the hot room was 7 to 8° and in the immediate post cold exposure it was 4°C (2° less than in the first test), the previous hot exposure effect still being evident.

1. Tissue Defect.

The requisite tissue defect for elimination of all cold-combatting powers is the complete destruction of the posterior hypothalamic gray. A section each from the series on Dogs 28 and 35 is shown in Figure 5 which illustrates such selectively placed lesions. The tissue defect must reach caudally to the cephalic border of the mammillothalamic tracts as they ascend from the mammillary bodies to penetrate the thalamic structures, dorsally to the ventral margin of the thalamus and laterally to approximate the medial aspects of the cerebrospinal tracts. There is no requisite cephalic border for the defect since a narrow caudal lesion severs all fibers traversing from higher levels. However, if the tissue defect involves the anterior hypothalamus sufficiently to attain a neurohypophysectomy, the thermo-regulatory picture is complicated by other factors. A deficit in the endocrine control of body water and a total absence of thirst are superimposed upon the uncomplicated neurogenic cold-combatting deficit described above. These additional deficits complicate the assaying of the status of heat regulating functions, but to what extent has not been elucidated other than the realization of their existence. These complications obtain both in the totally hypothalamectomized and in the anterior hypothalamectomized preparations.

B. Varying Degrees of Partial Impairment In Cold-combatting Powers.

Varying degrees of partial impairment of cold-combatting powers are illustrated in Figures 6 and 7. In Figure 6 the body cooling curves of Dogs 75, 96, and 112 are contrasted with that of Dog 28. Note that in Dog 75 the colonic temperature fell progressively in essentially a straight line, but more slowly than when a complete deficit obtained as in Dog 28. There was no detectable shivering. In Dog 112 the core temperature fell at the same rate as in 28 until it reached 31°C. At this point shivering became evident and the cooling curve changed considerably in slope although it continued downward. When the animal was removed from the 3°C environment and placed in a 23-25°C room colonic temperature rose

spontaneously and shivering continued until a body temperature of 31°C was again attained. These two dogs (75 and 112) possessed remnantal cold-combatting powers of a magnitude which is just detectable by this method of assay. Cold-combatting powers at this level could easily be missed by less exacting methods of evaluation.

A less severe cold-combatting deficit, yet a substantial one, obtained in Dog 96. Note that body temperature fell essentially in a straight line for the first few hours in spite of the presence of prominent shivering, but it then leveled off and even began rising again during the exposure period.

In contrast with the above major deficits the rectal temperature on Dog 36 illustrated a minor but interesting type of deficit. This animal exhibited a persistent 2°C hypothermia when housed at ordinary room temperature yet regulated essentially in a normal manner against a heavy cooling load over an 8 hour period. In order to raise rectal temperature to the normal 38°C level it was necessary to house it in a 28°C room. Subsequent to the 8 hour cold exposure body temperature returned immediately to the 36°C range.

Other types of lesser cold-combatting deficits are illustrated in Figure 7. In these experiments it was necessary to expose the animals to a heavy cooling load for several days, rather than for a few hours, to demonstrate a deficiency in their ability to resist exposure to cold. In Dog 48 the rectal temperature fell progressively in spite of prominent shivering, slowly for the 1st two days, then slightly more rapidly for 4 more days and during the 7th day considerably more rapidly. Dog 37 exhibited a somewhat similar but yet different deficit. It ran a mild hypothermia for five days and then during the sixth day of exposure the body temperature abruptly dropped from 36 to 31°C . Shivering was prominent during the entire exposure period.

A marginal deficit of considerable interest is the type shown by Dog 26. A hypothermia of 3°C developed immediately but was then maintained indefinitely (23 days) at this level when exposed to a heavy cooling load. Shivering was prominent during the time the 3°C hypothermia obtained.

1. Tissue Defect

When remnantal cold-combatting abilities were retained a small amount of posterior of hypothalamic gray tissue also remained intact. It matters not whether this be lateral, medial, dorsal or ventral hypothalamic gray tissue. When retained cold-combatting powers are greater than remnantal in magnitude a correspondingly

greater portion of hypothalamic gray tissue remains. The photographs in Figure 8 and 9 of sections taken from the series on Dogs 48 and 37 illustrate representative experiments. In Dog 48 only the left lateral and in 37 only the right dorsal hypothalamic gray remained intact.

If a relatively large portion of the posterior hypothalamic gray remains undisturbed, the animal's regulation against cold does not deviate materially from the normal. The photographs shown in Figure 10 are from the series of Dog 36. Note that a sizable portion of the posterior hypothalamic gray remained caudal to the tissue defect.

C. Regulation Against Warming Load.

The dogs which have the posterior hypothalamic gray selectively destroyed display no detectable deficit in regulating against moderate or heavy heating loads. This is illustrated in Figure 11 where the colonic temperature curves of Dogs 28 and 35 are contrasted with the response of an acute low mid-brain preparation when the animals were exposed to a hot environment. Only occasional bursts of panting occurred, meaning presumably that the dissipation of excess body heat was accomplished predominately by cardiovascular and body fluid adjustments. Note that the body temperature did not rise unduly during the exposure, i. e. the core temperature elevation was entirely within the normal response range for unoperated dogs. This was particularly true in Dog 28 where the rectal temperature did not rise above 39°C. There actually may have been a slight lowering of threshold in this instance.

The response of these poikilothermic-against-cold dogs to warming loads is quite in contrast to that displayed by low mid-brain preparations which retain no regulation against heat. In such an animal the body core temperature rises rapidly in a straight line and if it is allowed to rise above and/or to be maintained at 43°C for any appreciable length of time generalized irreversible physiological deterioration rapidly supervenes (heat prostration).

D. Ambulatory Preparations.

These animals proved to be ambulatory to the extent of not needing special nursing care other than the necessity of housing them in a warm environment. They were well oriented with their surroundings, ate and drank spontaneously, and maintained good nutrition and general physical condition.

E. Adverse Personality Changes.

Although ambulatory, the preparations did not prove to be as convenient laboratory tools as was hoped for because they exhibited a troublesome hyperirascibility. Ordinary handling precipitated furious bouts of rage which were without pseudo or sham aspects.

IV. DISCUSSION

It is fully realized that the criteria used herein for assaying the total absence of central cold-combating powers are only semiquantitative. The ultimate evaluation in order to be quantitatively absolute must involve oxygen consumption or equivalent determinations under basal conditions. Nevertheless, if preparations such as Dogs 28 and 35 retained any central heat production or heat conservation powers they were minor in extent and, if present, would not invalidate the conclusions drawn in this report.

An important problem is presented by preparations like Dog 75, illustrated in Figure 6, wherein there is no shivering yet their body temperatures fall more slowly than in the instances of the animals assumed to be totally poikilothermic. Is the slower fall in body temperature due to retained non-shivering heat producing ability or merely retained ability to restrict the loss of body heat? To answer this question oxygen consumption determinations (preferably direct calorimetry) will be necessary. This type of preparation would appear excellent for resolving the old question of possible sources of internal heat production other than by muscular activity, i. e. chemical versus physical heat production.

It is interesting that in this series there was no evidence of "release effects" as was encountered in the series alluded to previously. Elimination of all central regulation against cold was accomplished without detectably impairing central regulation against heat even to the extent of lowering or raising "panting and vasodilatation thresholds". This is clearly demonstrated in Dogs 28 and 35, and particularly so in Dog 28. The absence of "release effects" demonstrates that the "heat loss" mechanism, when properly separated from the "heat production" mechanism, is not even dependent on "reciprocal innervation" relationships.

The changes in personality encountered in these animals raised the question as to whether the anatomical basis for such changes was due to destruction of structural elements within the confines of the hypothalamus or to the functional involvement of structures distal to (bordering) the tissue defect. Sufficient anatomical material

is not as yet available to resolve this question. In this connection, it is to be recalled that since the early work of Head it has been appreciated that thalamic lesions produce changes in personality. Principally on this basis, but not entirely so, it is suspected that the adverse changes in personality, such as encountered in these experiments, are pathognomonic of associated thalamic involvement.

V. CONCLUSIONS

1. The neurogenic regulation of body temperature against a cool or cold environment is dependent upon neural elements located within the posterior hypothalamic gray.

2. Regulation against a warm or hot environment is not eliminated when the posterior hypothalamic gray is selectively destroyed.

3. Posterior hypothalamectomy does not eliminate the animal's orientation with its surroundings, its ability to eat and drink spontaneously, produce obvious gastrointestinal or nutritional deficits, or impair its motor or postural mechanism.

4. Such preparations are truly ambulatory but the presence of an increased susceptibility to rage limits their usefulness as laboratory tools.

VI. RECOMMENDATIONS

1. A continuing effort should be made to prepare poikilothermic dogs devoid of troublesome personality traits.

2. The localization of the neural elements responsible for regulation against heat should be further delimited.

3. The anatomical basis for the striking changes in personality encountered in the experiments should be systematically investigated.

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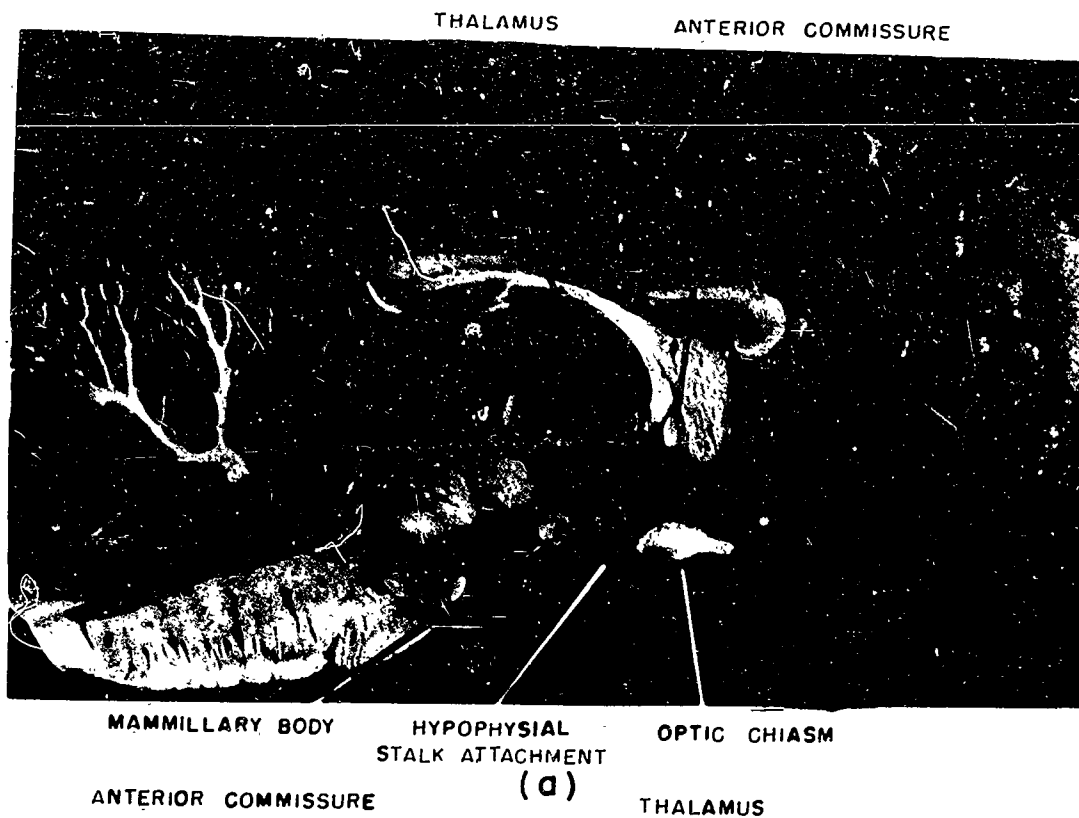
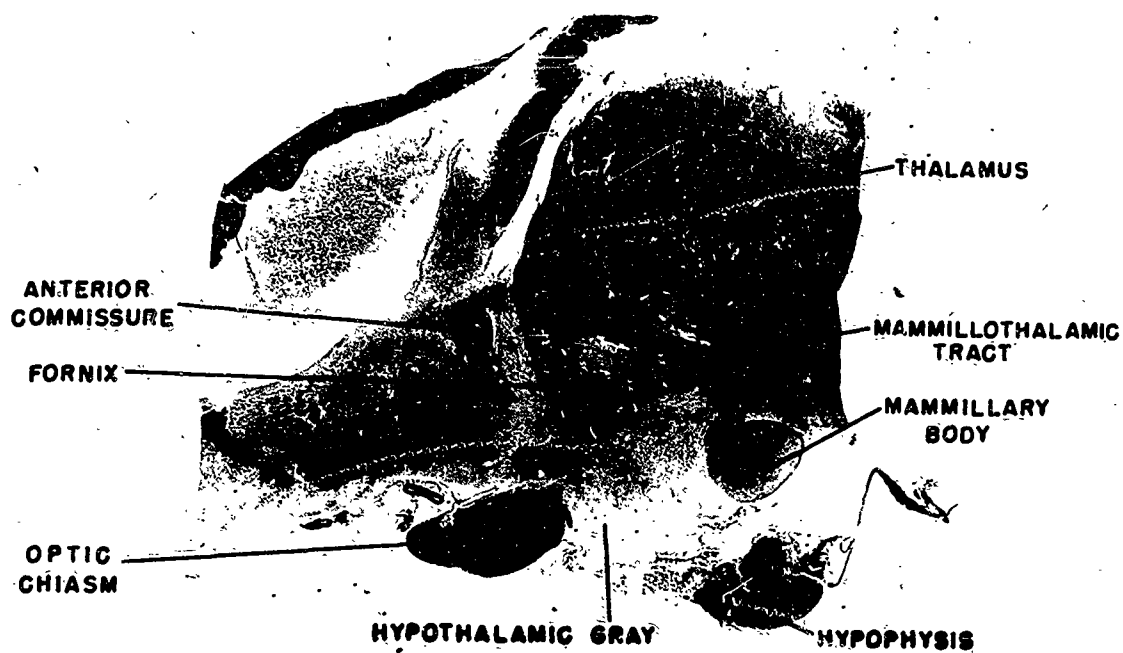


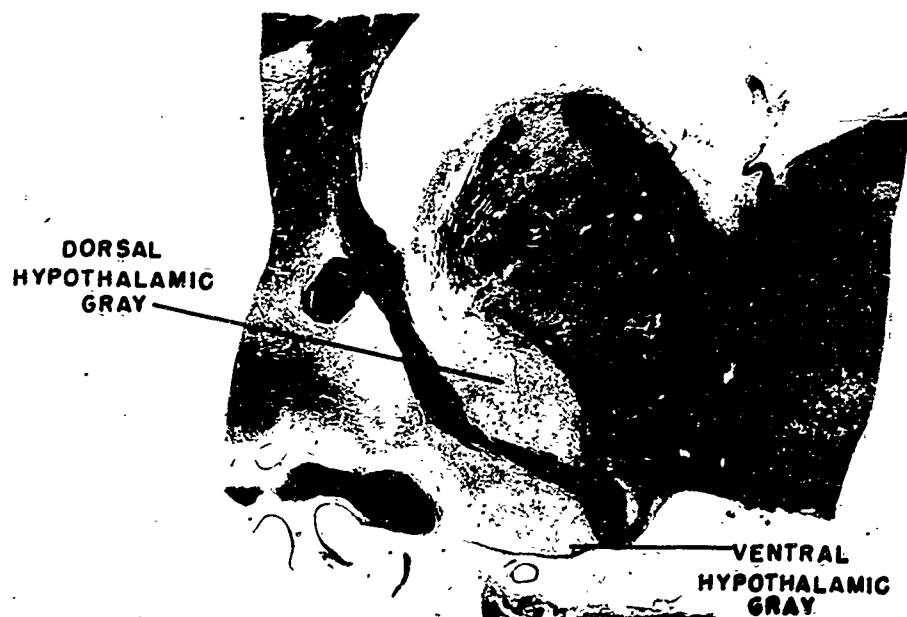
FIGURE 1.

a. A PHOTOGRAPH OF THE MEDIAL ASPECTS OF A DOG'S BRAIN CUT SAGITTALLY ALONG THE MIDLINE. NOTE THE LOCATION AND RELATIVE SIZE OF THE HYPOTHALAMUS WITH RESPECT TO OTHER MAJOR STRUCTURAL ENTITIES.

b. A PHOTOGRAPH OF A DOGS BRAIN DEMONSTRATES A PRACTICE COAGULATION OF THE POSTERIOR HYPOTHALAMUS. NOTE THAT THE MIDBRAIN, THALAMUS, ANTERIOR HYPOTHALAMUS AND HYPOPHYSIS REMAINED UNDISTURBED.



(a)



(b)

FIGURE 2.

a. A PAL-WEIGERT SECTION THROUGH THE PLANE OF A MAMMILLOTHALAMIC TRACT. NOTE HOW SHARPLY THE HYPOTHALAMIC GRAY IS DEMARCATED FROM NEIGHBORING STRUCTURES.

b. A PAL-WEIGERT SECTION THROUGH THE PLANE OF A FORNIX SHOWS THE DIVISION OF THE HYPOTHALAMIC GRAY INTO DORSAL AND VENTRAL COMPONENTS. NOTE AGAIN THE CLEAR DELINEATION OF THE HYPOTHALAMIC GRAY.

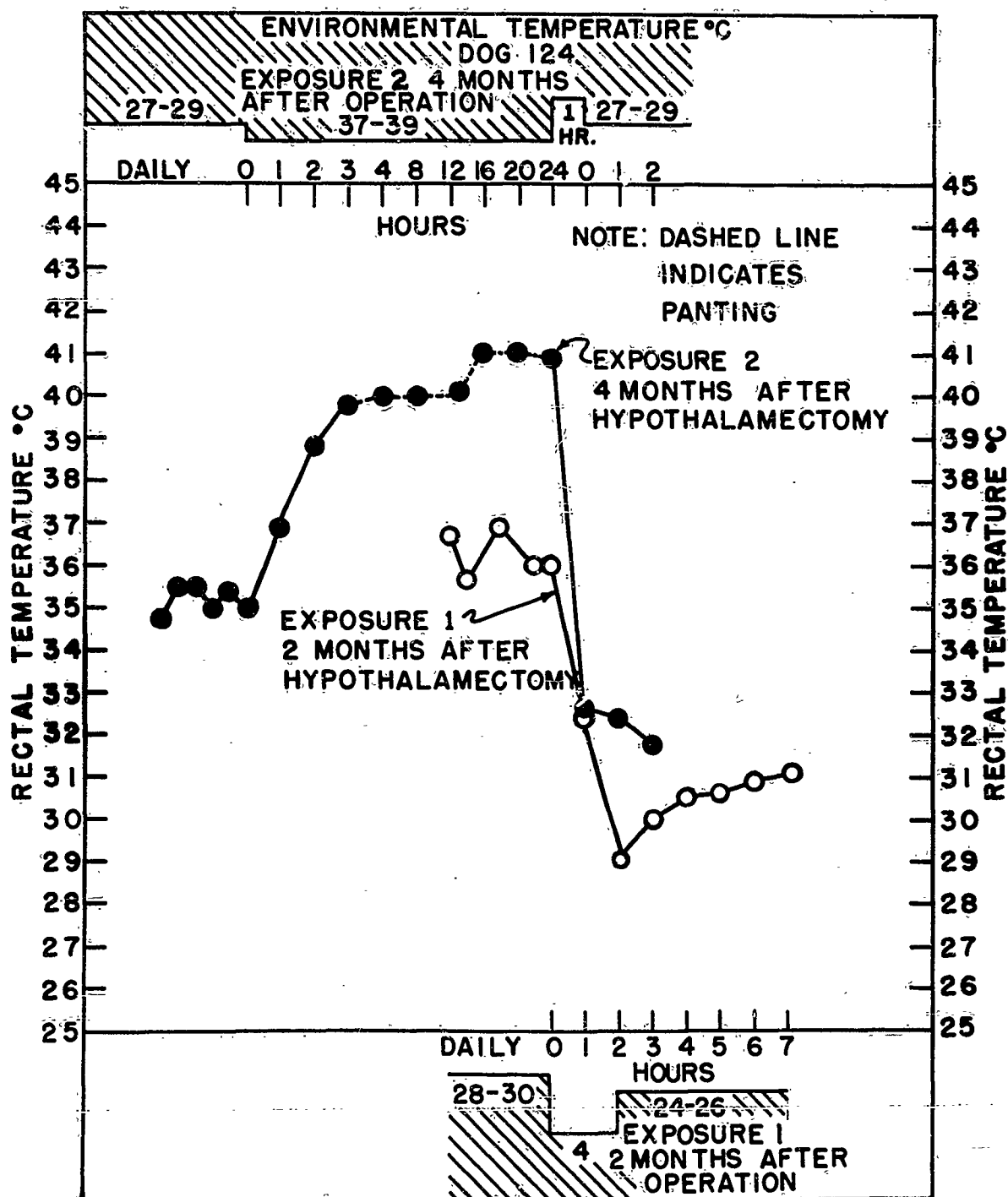


FIGURE 4 SEE TEXT FOR DETAILS.

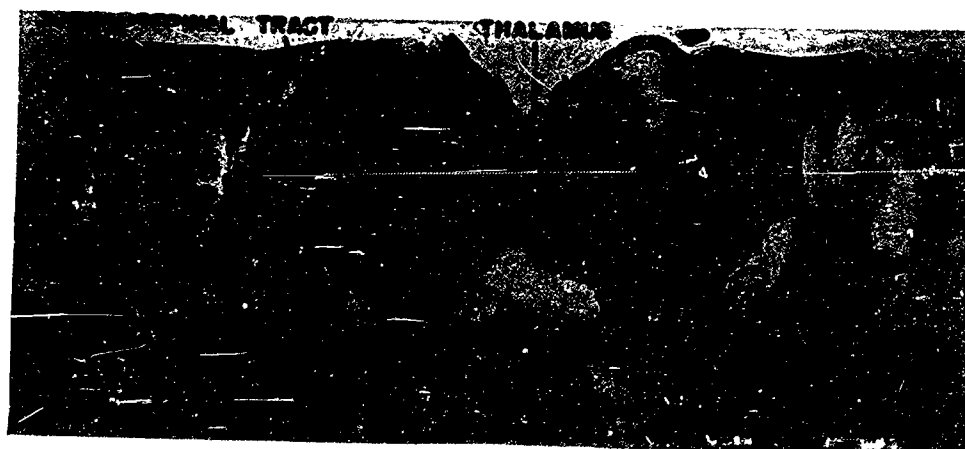
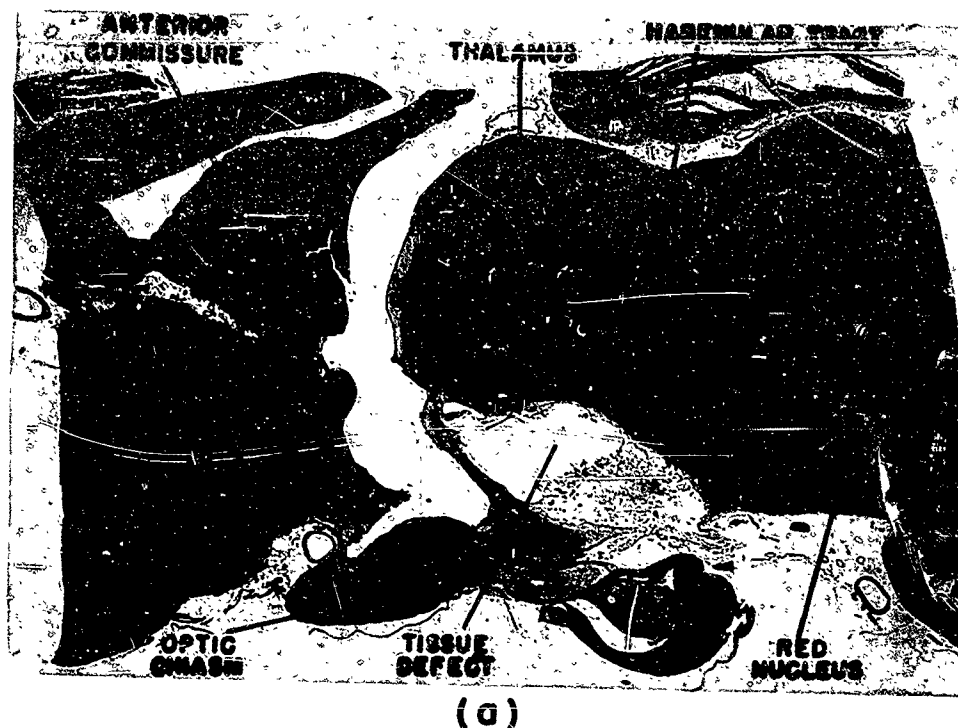


FIGURE 5.

a. A PAL-WEIGERT SAGITTAL SECTION TAKEN FROM THE SERIES ON DOG 28.

NOTE: (1) THE TISSUE DEFECT ENCOMPASSES THE ENTIRE POSTERIOR HYPOTHALAMIC GRAY.

(2) THE TISSUE DEFECT IS NOT FILLED WITH SCAR TISSUE BUT HAS BECOME A COMPONENT OF THE VENTRICULAR SYSTEM.

(3) THE NEIGHBORING TISSUE IS NOT DISTURBED INCLUDING THE ANTERIOR HYPOTHALAMUS AND HYPOPHYSIS.

b. A PAL-WEIGERT CROSS SECTION TAKEN FROM THE SERIES ON DOG 35.

NOTE: (1) THE HYPOTHALAMIC TISSUE DEFECT EXTENDS Laterally TO APPROXIMATE THE MEDIAL ASPECTS OF THE CEREBROSPINAL TRACTS.

(2) ANEMIC INFARCTS DORSALLY IN THE THALAMUS.

(3) THE HYPOPHYSIS IS NOT DISTURBED.

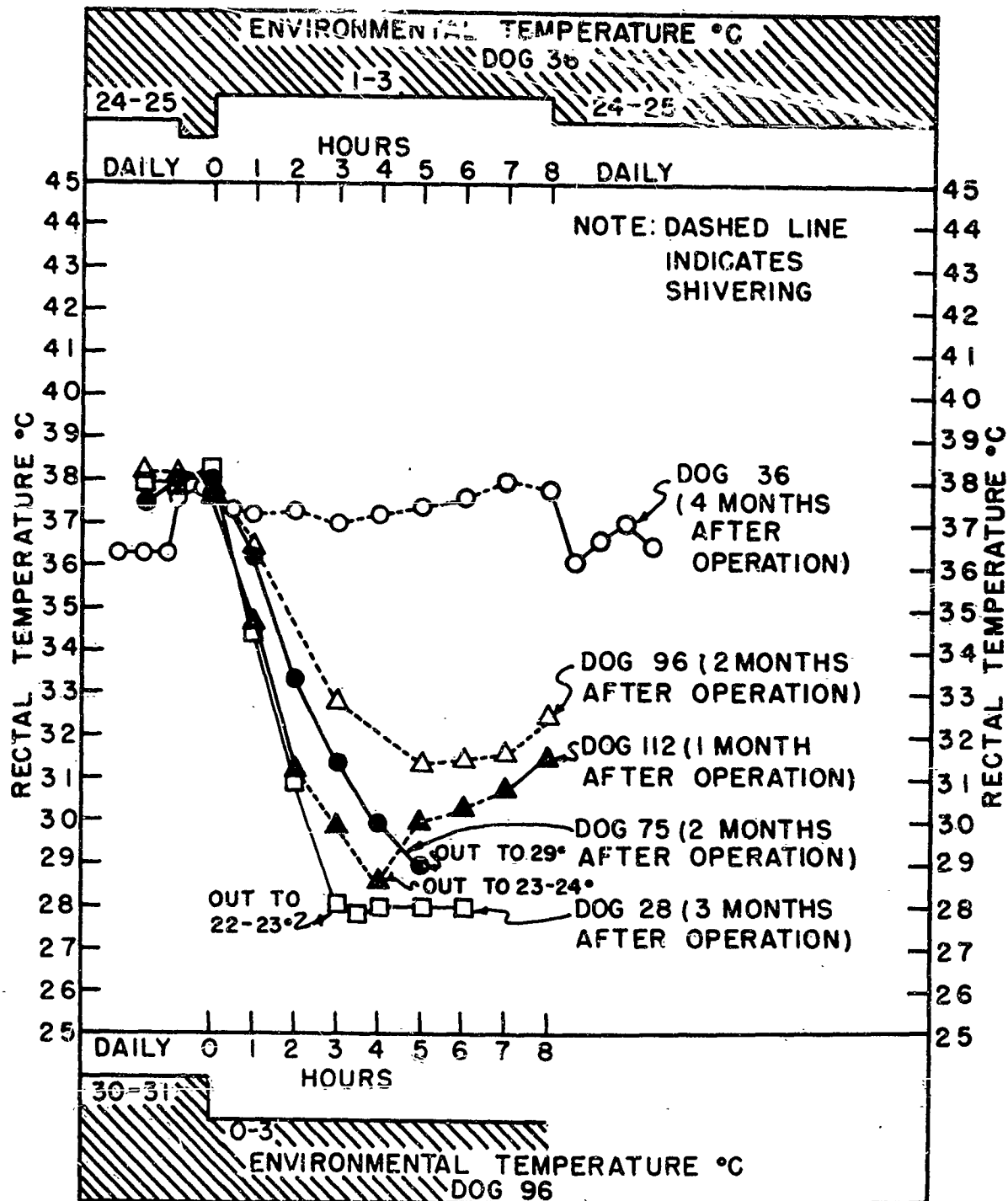


FIGURE 6 SEE TEXT FOR DETAILS.

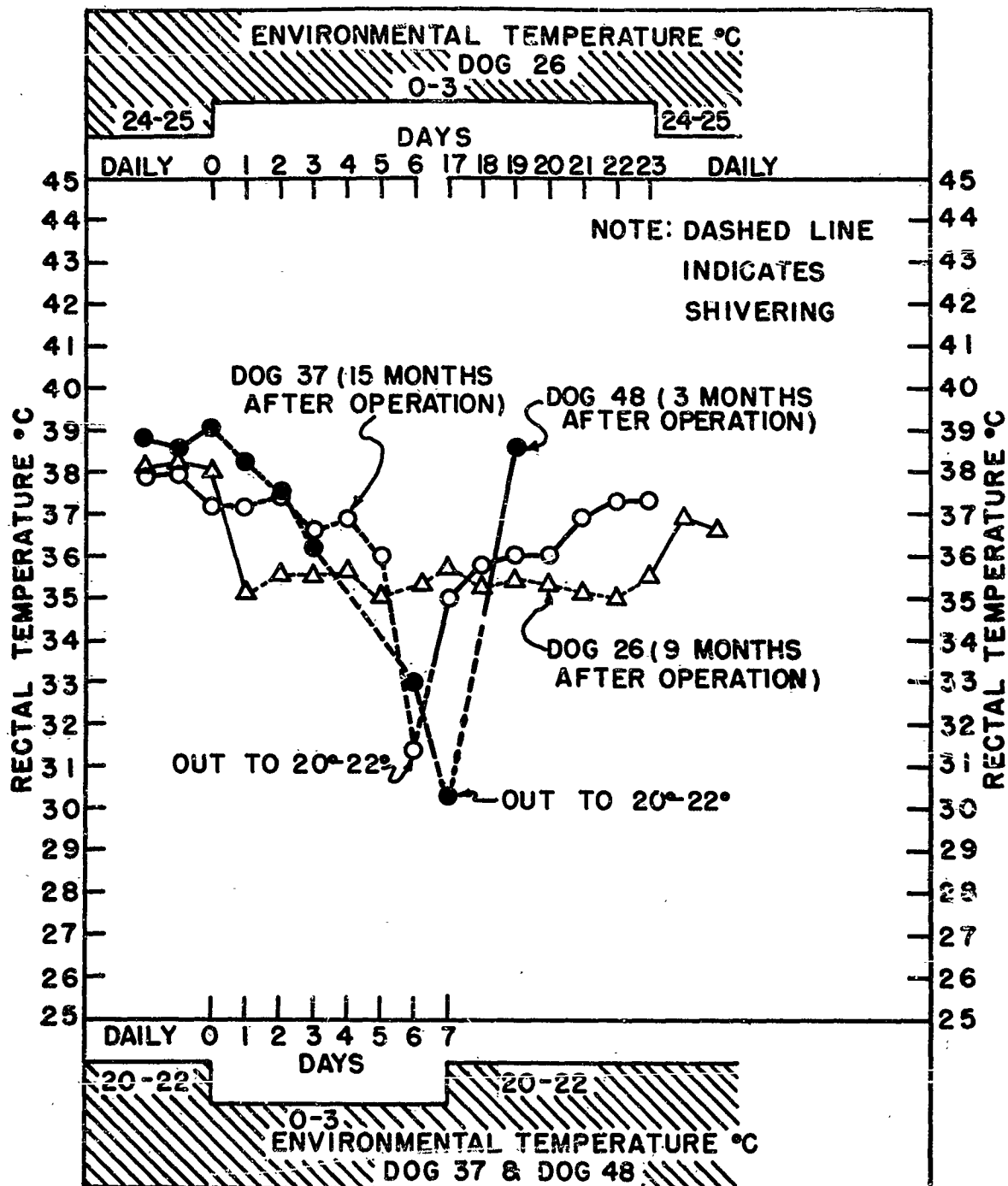
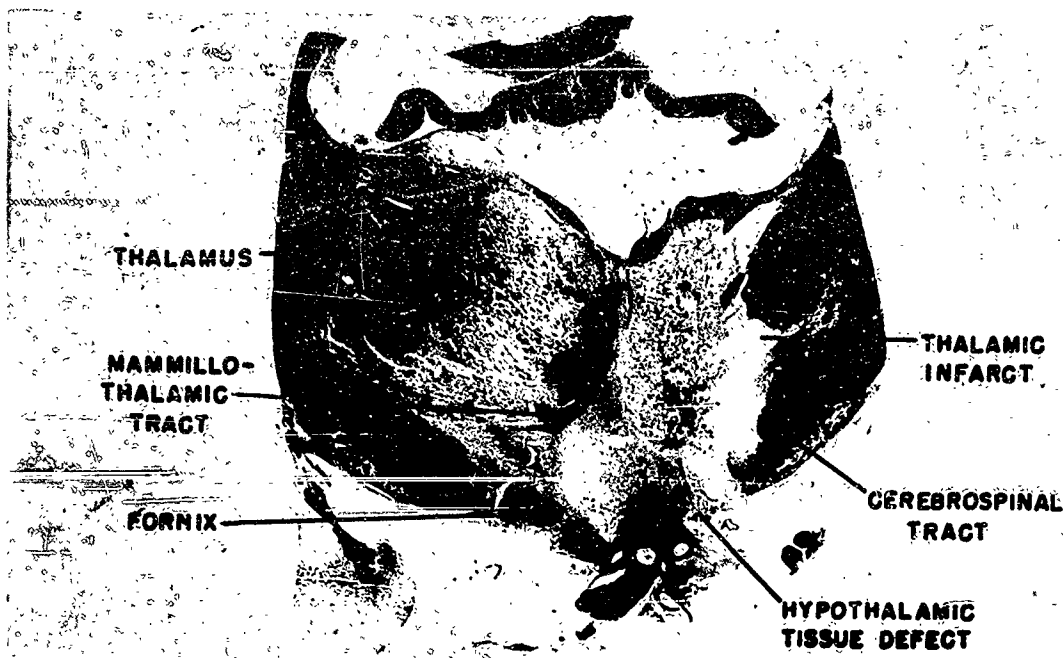
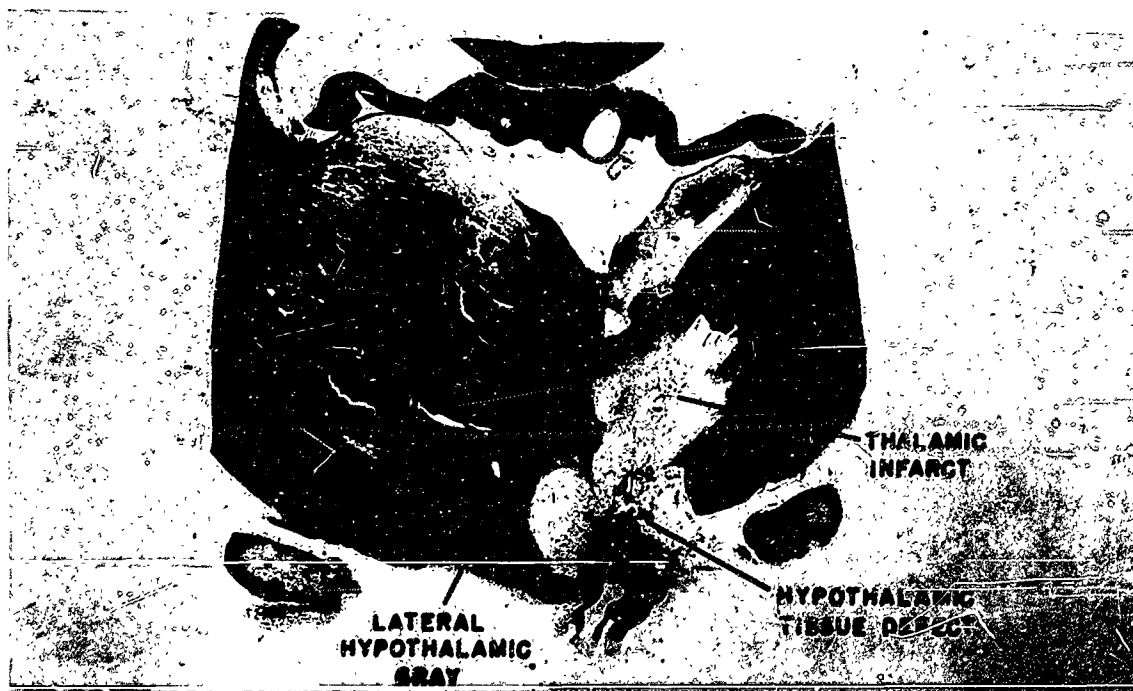


FIGURE 7 SEE TEXT FOR DETAILS.



(a)



(b)

FIGURE 8-

PAL-WEIGERT CROSS SECTIONS TAKEN FROM THE SERIES ON DOG 48. NOTE THAT THE RIGHT LATERAL AND BOTH MEDIAL COMPONENTS OF THE HYPOTHALAMIC GRAY WERE SELECTIVELY DESTROYED AND THAT ONLY THE LEFT LATERAL (LATERAL TO MAMMILLOTHALAMIC TRACT AND FORNIX) HYPOTHALAMIC GRAY REMAINED. THIS DOG EXHIBITED ONLY A MARGINAL DEFICIT IN REGULATION AGAINST COLD, SEE FIGURE 7.

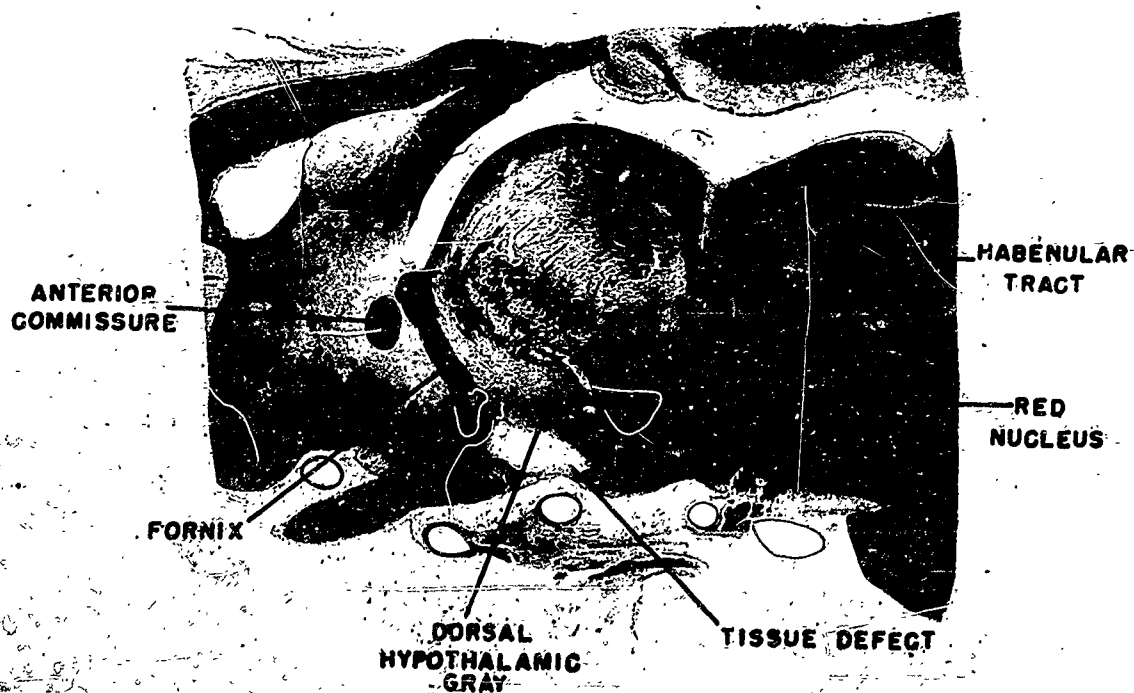
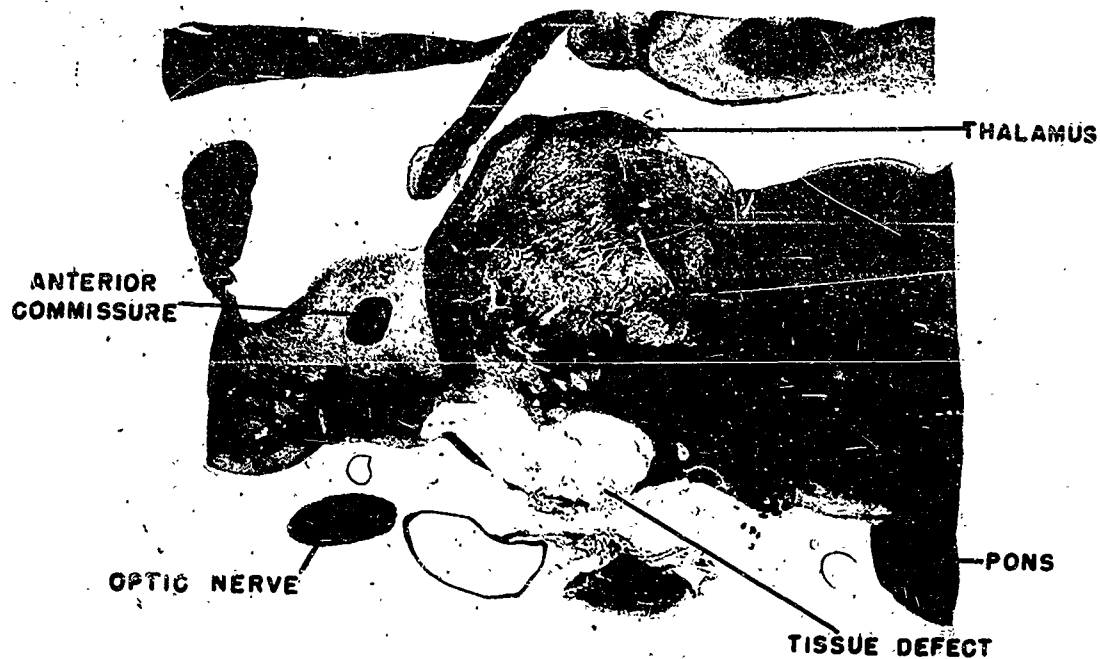


FIGURE 9. PAL-WEIGERT SAGITTAL SECTIONS TAKEN FROM THE SERIES ON DOG 37
 NOTE: ONLY THE RIGHT DORSAL HYPOTHALAMIC GRAY REMAINED INTACT.
 THIS DOG EXHIBITED ONLY A MARGINAL DEFICIT IN ITS REGULATION
 AGAINST COLD, SEE FIGURE 7.

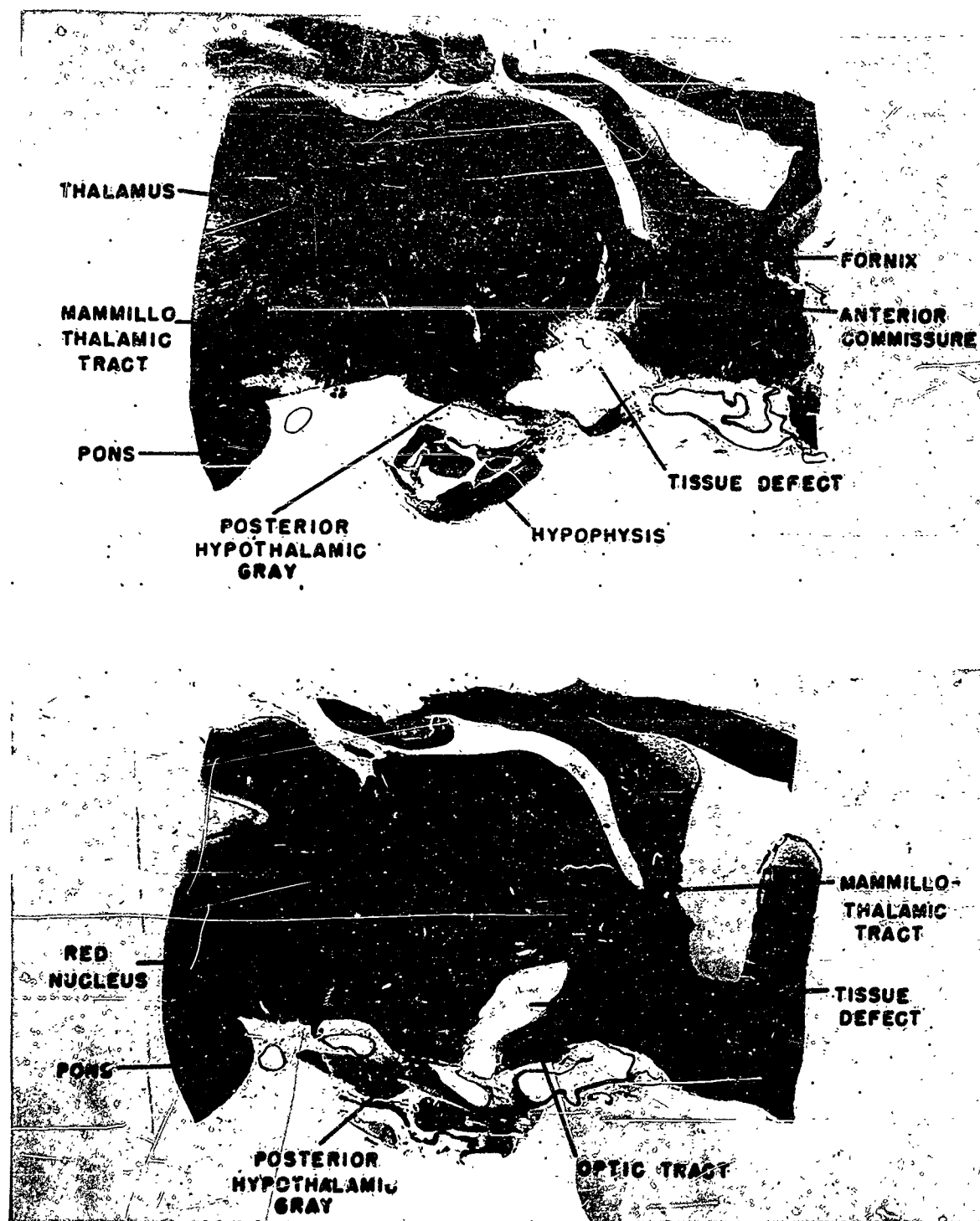


FIGURE 10 - PHOTOGRAPH OF SAGITTAL SECTIONS TAKEN FROM PAL-WEIGERT SERIES ON DOG 36.

NOTE: THE TISSUE DEFECT INVOLVES A LARGE PORTION OF THE HYPOTHALAMIC GRAY LEAVING ONLY APPROXIMATELY ONE HALF OF THE POSTERIOR HYPOTHALAMIC TISSUE INTACT. THIS DOGS REGULATION AGAINST COLD AND AGAINST HEAT WAS ESSENTIALLY NORMAL.

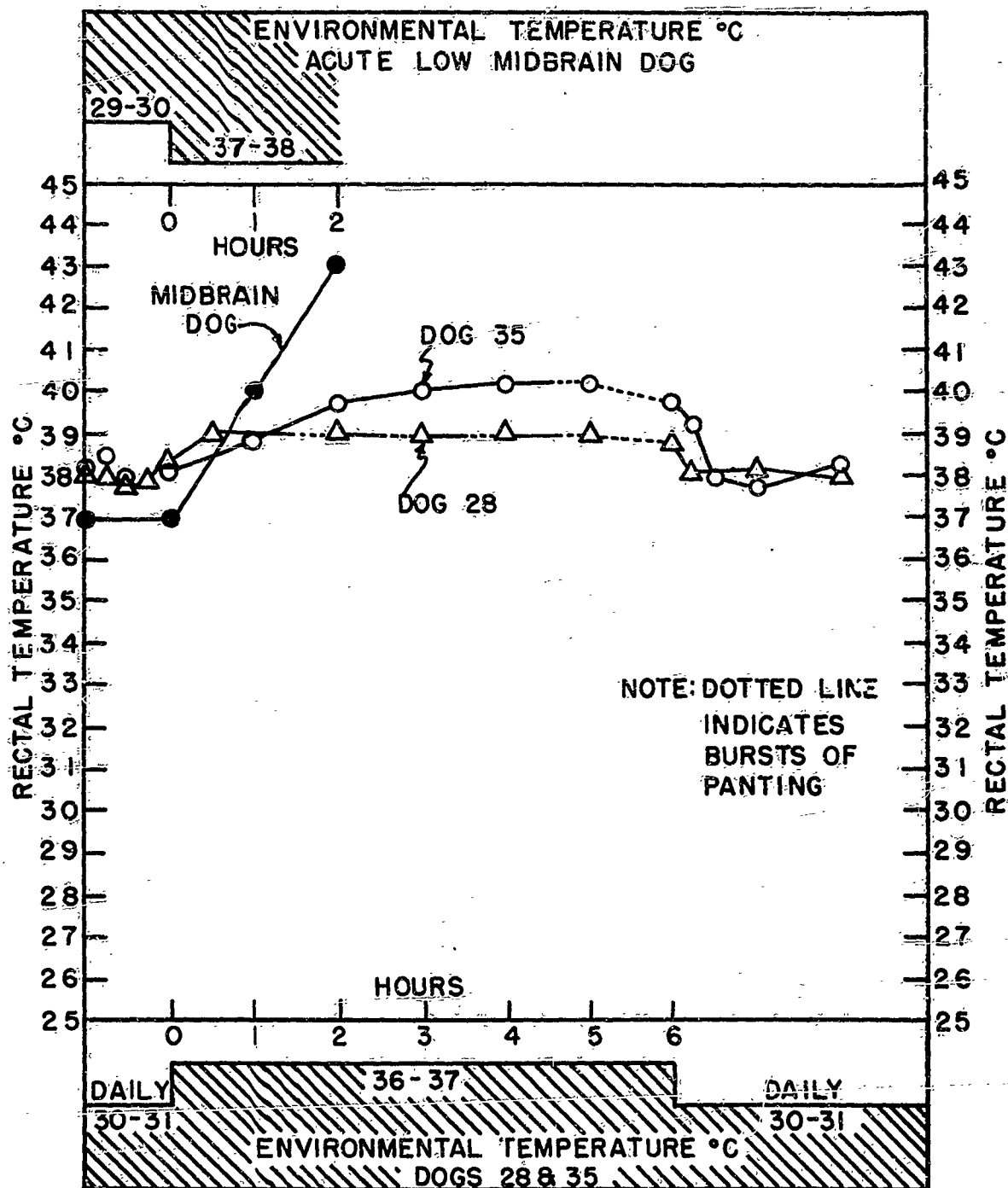


FIGURE II SEE TEXT FOR DETAILS.

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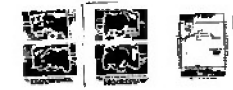
THE AMBULATORY SEMI-POIKILOTHERMIC DOG (POIKILOTHERMIC AGAINST COLD) - AMRL PROJECT NO. 6-64-12-028, by Allen D. Keller, and Henry Batsel. 10 June '52, 26 pp. incl. photos, graphs.

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